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Editorial Comment

Expanding Indications for Radiofrequency Catheter Ablation: Ventricular Tachycardia in Association With Right Ventricular Dysplasia?

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Radiofrequency catheter ablation has become a first-line curative therapy for the majority of the common forms of supraventricular tachycardia, and for some forms of ventricular tachycardia (VT). For example, VT occurring in the absence of structural heart disease such as idiopathic left VT or right ventricular outflow tract tachycardia can now be cured by radiofrequency catheter ablation with a high degree of efficacy and safety (1–6). In contrast, ablation of VT in patients with underlying structural heart disease has not yet been widely accepted as safe and effective. In this issue of the Journal, Ellison et al. (7) expand on the role of radiofrequency catheter ablation for the treatment of VT with a report on their clinical experience using entrainment pace mapping to guide radiofrequency catheter ablation of VT in patients with arrhythmogenic right ventricular dysplasia (ARVD). The authors describe a small series of five patients who presented with sustained, symptomatic VT. None of the patients had a history of syncope or ventricular fibrillation. The diagnosis of ARVD was based on the presence of electrocardiogram abnormalities, right ventricular enlargement and/or hypokinesis on echocardiogram and inducible VT with a left bundle branch block

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pattern. A total of 19 morphologically different VTs were induced. Pacing entrainment was performed from 58 sites in a manner similar to that previously described by the authors and others for mapping and ablation of VT in patients with ischemic VT. The VT was noted to terminate during ablation at 13 of 58 sites, mostly sites in the inferior-basal right ventricle near the tricuspid annulus or in the right ventricular outflow tract. In all, 8 of 19 VTs were ablated (i.e., terminated and not reinducible) without complications. Although four of five patients still had inducible VT after ablation, it appears from the results that none has had a spontaneous recurrence of VT in follow-up (mean 17 months) despite the fact that four of five patients were previously refractory to one or more antiarrhythmic drugs. During follow-up, one patient was off all treatment, three patients were on antiarrhythmic drugs (primarily amiodarone) and one had an implantable cardioverter defibrillator (ICD) implanted. Thus, at first glance the results of ablation appear encouraging. However, as the authors themselves point out, this is a small series of patients with a relatively short follow-up period. Consequently, the overall acute and long-term safety and efficacy of radiofrequency catheter ablation for VT in patients with ARVD may not be accurately represented by this study.

The authors' primary emphasis in this report, as suggested by the title, was that VT in patients with ARVD shares many features with VT in patients with ischemic heart disease and myocardial infarction (8,9). For example, on the basis of responses to entrainment, the VT in these five patients with AVRD was determined to be due to reentry. Furthermore, specific sites of interest in the VT reentrant circuit were identified by entrainment (e.g., exit sites, central or proximal areas of slow conduction, bystander sites, etc.) and used to guide radiofrequency catheter ablation. However, the authors' conclusion that entrainment can actually characterize the anatomic and electrophysiologic nature of the VT reentry circuits in ARVD may not be entirely supported by their data. Such a complete characterization of the reentry circuits, as suggested in their Figure 1, would probably require much more extensive and detailed mapping than performed in this study. This may explain in part the relative inaccuracy of entrainment in predicting the efficacy of radiofrequency catheter ablation at various sites in the VT reentry circuits in these patients. For example, at exit and central/proximal sites where efficacy would be expected to be high, only 25% and 50% of energy applications terminated VT, respectively. Furthermore, ablation at outer loop and adjacent bystander sites, where efficacy might be expected to be low, terminated VT during 25% of energy applications. Thus, the relative inaccuracy of entrainment-guided ablation in ARVD suggests that the VT reentrant circuit may be more complex than shown in Figure 1. This should not be surprising considering the complex and unpredictable nature of the fibro-fatty infiltration of myocardium in ARVD (10–13). It is also possible that epicardial reentry may account for failure of ablation from the endocardial surface in some cases.

Nonetheless, the fact that the authors have safely and successfully mapped and ablated VT in some patients with ARVD suggests yet another potential indication for radiofrequency catheter ablation. Similar success has been reported by others, further supporting the authors' observations (14–17). However, the authors correctly acknowledge that the small number of patients in their study is insufficient to assess reasonably the risk of procedural complications and the long-

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term efficacy of ablation in patients with ARVD. These issues must be carefully addressed, perhaps in the form of a large multicenter trial or national registry, to delineate the role of radiofrequency catheter ablation in the treatment of VT in ARVD.

With respect to the risk of procedural complications, the thinning of the right ventricular myocardium and replacement with fibro-fatty infiltrates in ARVD raises concerns regarding the possibility of perforation and pericardial tamponade during ablation. There was no report of perforation in this study, but the number of patients studied was small, as the authors appropriately point out. The authors did not note the use of magnetic resonance imaging (MRI) to determine the extent of dysplasia in their patients. However, this diagnostic tool may be of some value in the diagnosis of ARVD and in qualitative assessment of the extent of dysplasia and thinning of the right ventricle (18–20). Such information may be useful in determining the safety of ablation vs. medical or device therapy for VT in patients with ARVD. Certainly in those patients with severe right ventricular dysplasia and thinning on MRI, it may be prudent to consider antiarrhythmic drug therapy (e.g., amiodarone) or ICD implantation in lieu of ablation to avoid possible procedural complications.

Of additional concern is the fact that patients with ARVD may present anytime in the course of their disease with syncopal VT or arrhythmic sudden death (21–32). Thus, while the patients in this study had no history of syncope or aborted sudden death, there is no assurance that they will not present with a more serious or life-threatening episode of VT in the future. Furthermore, since ARVD is typically a progressive disease (10–13,33), new VTs could develop in the future despite apparently successful ablation at present.

Finally, in this study the majority of patients were continued on antiarrhythmic drugs therapy with amiodarone or had an ICD implanted following ablation. Thus, ablation was actually used as a palliative or adjunctive treatment as it is usually used in ischemic VT (8,9). Therefore, although the authors have shown the feasibility of mapping and ablating VT in patients with ARVD, ablation as sole therapy should probably not be condoned without a controlled trial comparing it with standard antiarrhythmic drug (e.g., amiodarone) or device (i.e., ICD) therapy to demonstrate at least equivalent safety and efficacy.

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